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REVIEW ARTICLE

The anatomical basis of upper limb dystonia: lesson from secondary cases

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Abstract Upper limb dystonia is a focal dystonia that may affect muscles in the arm, forearm and hand. The neuroanatomical substrates involved in upper limb dystonia are not fully understood. Traditionally, dysfunction of the basal ganglia is presumed to be the main cause of dystonia but a growing body of evidence suggests that a network of additional cortical and subcortical structures may be involved. To identify the brain regions that are affected in secondary upper limb dystonia may help to better understand the neuroanatomical basis of the condition. We considered only patients with focal upper limb dystonia associated with a single localized brain lesion. To identify these patients, we conducted a systematic review of the published literature as well as the medical records of 350 patients with adult-onset dystonia seen over past 15 years at our movement disorder clinic. The literature review revealed 36 articles describing 72 cases of focal upper limb dystonia associated with focal lesions. Among patients at our clinic, four had focal lesions on imaging studies. Lesions were found in multiple regions including thalamus ($n = 39$), basal ganglia ($n = 17$), cortex ($n = 4$), brainstem ($n = 4$), cerebellum ($n = 1$), and cervical spine ($n = 7$). Dystonic tremor was not associated with any particular site of lesion, whereas there was a trend for an inverse association between task specificity and thalamic involvement. These data in combination with functional imaging studies of idiopathic upper limb dystonia support a model in which a network of different regions plays a role in pathogenesis.

Keywords Upper limb dystonia · Brain damage · Task specificity · Tremor

Introduction

Upper limb dystonia (ULD) is a focal dystonia with variable clinical expression. Dystonic overactivity may involve muscles in the arm, forearm and hand, and dystonic tremor may also be present [1]. An interesting feature that distinguishes ULD from other focal dystonias is task specificity, namely, when dystonia affects one task alone, like writing, playing a musical instrument, etc. [1–3]. However, task specificity may be lost with time and ULD tends to manifest during several non-specific action or spontaneously [4]. It is also possible that ULD presents initially as a non-task specific dystonia [5].

The anatomical basis of ULD is poorly known. The traditional view commonly holds that ULD, like other forms of dystonia, is due to a basal ganglia disorder. However, recent voxel-based morphometry (VBM) studies in idiopathic ULD have shown microstructural gray matter (GM) changes not only in the basal ganglia but also in the sensory-motor cortex, thalamus and cerebellum [6]. It remains unclear whether GM changes in these brain regions reflect the trigger for ULD or are merely downstream consequences of the clinical disorder. Whether the facultative presence of associated dystonic tremor and task specificity reflect involvement of particular anatomic areas also remains to be clarified.

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To identify the brain regions that are affected in secondary ULD might help to better delineate the anatomical basis of the condition. This issue has received little attention because focal lesions causing ULD are uncommon and most information comes from case reports or small case series. We, therefore, reviewed neuroimaging findings in patients with presumed secondary ULD associated with a single identifiable focal lesion on neuroimaging studies, including a group of unpublished cases from our movement disorder clinic.

Methods

A computer-assisted review of the literature using PubMed as the search engine was performed to identify relevant articles. The search was run from January 1950 to February 2015 using movement "disorders", "upper limb dystonia", "hand dystonia", and "post-hemiplegic dystonia" as key words alone or in combination with terms like "brain lesion" and "secondary". By application of predefined criteria, articles had to be original full texts and describe patients with focal ULD carrying a unique focal CNS lesion on neuroimaging studies. ULD was considered as task-specific dystonia when dystonia was always triggered by the same activity. Non-task-specific ULD was diagnosed if dystonia occurred predictably during several non-specific tasks, or spontaneously.

Exclusion criteria were: ULD as part of a segmental/multifocal or generalized dystonia; ULD developing in the setting of a neurodegenerative disorders, or after exposure to neuroleptic drugs; ULD associated with multiple localized brain lesions or with lesions that could not be localized (such as those with hypoxia/ischemia or diffuse atrophy) on computed tomography (CT) scan/magnetic resonance imaging (MRI) study.

A total of 36 articles (published from 1955 to February 2015) [6–42] reporting 72 cases of presumed secondary focal ULD were found. Each article was reviewed to extract, if possible, information on sex, age at ULD presentation, type and location of brain lesion, age at imaging study, task specificity of ULD, presence of associated tremor or other neurological features.

After obtaining approval from the institutional review board, medical records of 350 patients seen between January 2001 and February 2015 at the movement disorder clinic of the University of Bari, Italy, were also reviewed. Inclusion and exclusion criteria as well as collected data were the same we used to assess patients from the literature.

Statistical analysis was performed by the Stata 11.0 package (Stata Corporation, College Station, TX). Data were expressed as percentages of patients, unless otherwise indicated. Differences across groups were analyzed by Fisher test (two tailed) or χ^2 test, as appropriate.

Results

The overall study group included 76 patients, 72 from the literature [7–42] and 4 from our movement disorder clinic (Table 1). In the overall group, stroke was the most common etiology [7, 9–24, 27–30] ($n = 60$), followed by brain tumors [15, 25, 31, 34, 40] ($n = 5$) demyelinating lesions [35, 36] ($n = 3$), syringomyelia [37] ($n = 2$), traumatic injury [32, 38] ($n = 2$), vascular malformation [26] ($n = 2$), brain infectious [33] ($n = 1$) and calcification [8] ($n = 1$). ULD developed acutely in nine patients [10, 14–16, 29, 35, 36, 41]. The average age at ULD presentation was 51.7 years (SD 17.7, range 9–90 years) and the average disease duration was 6.5 years (SD 9.8). Sex was not specified in seven cases [24]. In the remaining 69 patients women predominated (n 40). Dystonic tremor was observed in 18 patients (24 %) [11–13, 19, 21, 23, 28, 29, 39]. In most cases (n 70/76, 92 %), ULD presented initially as a non-task-specific dystonia, whereas task specificity

Table 1 Clinical features of the four patients from our clinic

Patients	1	2	3	4
Sex	Male	Female	Female	Male
Age at onset of dystonia (years)	59	57	46	72
Disease duration (years)	11	18	33	4
Topography and phenomenology of dystonia	Right hand dystonia, non task specific	Right hand dystonia, writer's cramp	Left hand dystonia, non task specific	Right hand dystonia, writer's cramp
Cause of dystonia	Infarction	Infarction	Infarction	Infarction
Time elapsing from brain lesion to dystonia onset	Unknown	Unknown	Unknown	Unknown
Site of brain lesion	Left caudate/putamen	Right midbrain	Right caudate	Left putamen
Associated neurological signs	None	None	None	None

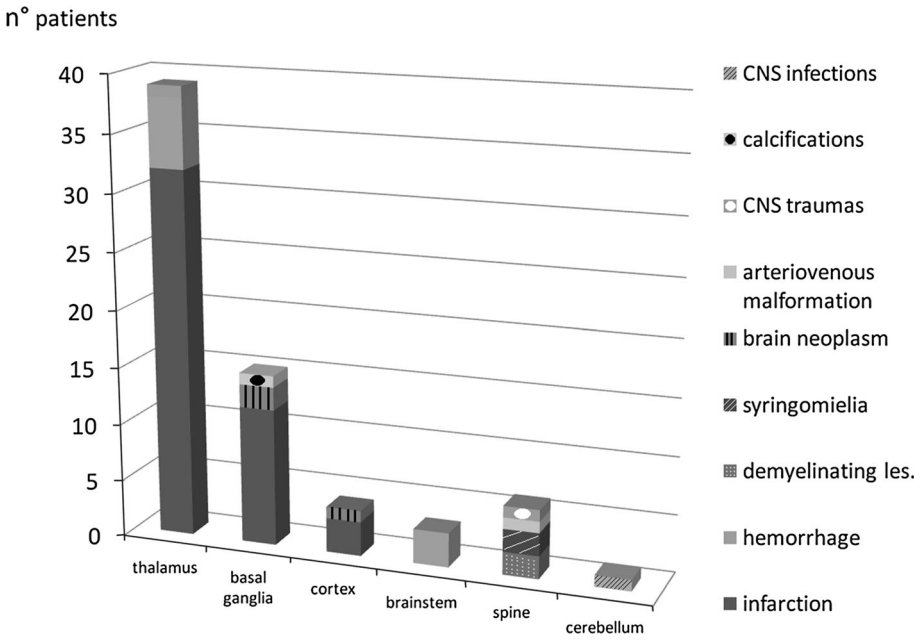

Fig. 1 Site and etiology of brain lesions in patients with secondary upper limb dystonia

was reported in only 6/76 patients (8 %), 2 from our clinic and 4 from the literature [25, 27]. Associated symptoms included hemiparesis (*n* 44/76, 58 %) [7, 9, 12, 13, 16, 17, 19–24, 27] and hemipoesthesia (*n* 13/76, 17 %) [12, 20–22, 24] either alone or in combination. There was a non-significant trend to a lower frequency of hemiparesis in patients with task-specific ULD (1/6 vs. 41/70, $p = 0.084$), while there was no difference in sensory impairment between patients with task-specific ULD and those with non-task-specific ULD (2/6 vs. 19/70, $p = 0.7$).

Head imaging study was performed by MRI in 48 patients, by CT scan in 28 patients. As presumed in Fig. 1, the most commonly affected region was the thalamus ($n = 39/76$, 51 %) [7, 12, 13, 15, 17–21, 24, 27], especially the posterior lateral regions ($n = 32$). The basal ganglia was the next most commonly affected region ($n = 17/76$, 22 %) [8–13, 15, 20, 23, 26], with four in the caudate [8, 13, 15], four in the putamen [9–11, 23, 27], and nine in both caudate and putamen [9, 12, 13, 20]. Four additional patients (5.3 %, one from our series) had lesions in the brainstem (two in the midbrain, two in the pons) [14, 16, 23]. The frontal cortex was affected in two patients (2.6 %) [23], and the parietal cortex in four (5.3 %) [22, 25]. Finally, one patient (1.3 %) had lesion in the cerebellum and seven patients (9.2 %) in the posterior region of the cervical spine. Brain lesion was right-sided in 35 patients, left-sided in 41. Patients with lesions localized in the cortex, basal ganglia, of thalamus developed contralateral dystonia, whereas patients with lesions localized in the brainstem, spinal cord, or cerebellum developed ipsilateral dystonia. Regional distribution of lesions did not differ between patients who performed MRI and those who underwent CT scan of the head (data not shown, $p = 0.54$).

There was no significant association between dystonic tremor and any site of lesion (Table 2), even when we considered only the 32/39 patients who had posterior lateral thalamic damage. Table 3 shows that there was a non-significant trend for an inverse association between task specificity and thalamic involvement ($p = 0.052$). When we compared patients with lesions in the thalamus and

Table 2 Distribution of brain lesions in patients who had dystonia and tremor in the upper limb and patients who had upper limb dystonia alone

Site of brain lesion	Patients who had dystonia and tremor in the upper limb dystonia (*n*. 16)	Patients who had upper limb dystonia without associated tremor (*n*. 60)
Basal ganglia	4 (25 %)	15 (25 %)
Thalamus	11 (73 %)	28 (47 %)
Cortex	1 (2 %)	5 (8 %)
Brainstem	0	4 (7 %)
Spine	0	7 (11 %)
Cerebellum	0	1 (2 %)

(Chi square test, 2 × 6: $p = 0.53$)

Table 3 Distribution of brain lesions in patients who had task specific and non task specific upper limb dystonia

Site of brain lesion	Patients who had task specific upper limb dystonia (*n*. 6)	Patients who had non task specific upper limb dystonia (70)
Basal ganglia	2 (33 %)	17 (24 %)
Thalamus	0	39 (56 %)
Cortex	2 (33 %)	4 (5.7 %)
Brainstem	1 (17 %)	3 (4.3 %)
Spine	1 (17 %)	6 (8.6 %)
Cerebellum	0	1 (1.4 %)

(Chi square test, 2 × 6: $p = 0.052$)

patients with lesions in other CNS sites, we observed a significant inverse association between task specificity and thalamic involvement (task specific ULD: 0/39 patients with thalamic involvement vs. 6/37 patients with involvement of other CNS sites; $p = 0.014$). This association remained significant ($p = 0.03$) even when we included the 32/39 patients with posterior lateral thalamic damage. Similar findings were obtained considering only patients who performed MRI (data not shown).

Discussion

Our review of cases who developed ULD after a single focal brain lesion showed that secondary ULD may be associated with lesions in several CNS areas including frontal–parietal cortex, thalamus, basal ganglia, cerebellum, brainstem, and cervical spine. Stroke was the most frequent etiology in this sample, which probably explains the greater number of lesions in the thalamus and basal ganglia, locations that are frequently targeted by vascular insults. Of note, lesions in the cortex, thalamus and basal ganglia were contralateral to ULD, whereas lesions in the cerebellum, brainstem and cervical spine were ipsilateral to ULD. In most patients, secondary ULD presented initially as a non-task-specific dystonia, whereas task specificity was observed in less than 8 % of patients. An interesting finding from our analysis was the association between task specificity and lack of thalamic involvement. Conversely, dystonic tremor was not associated with any particular site of lesion.

Although it is not possible to rule out the occurrence of microstructural defects or functional disturbances in other brain regions that appear grossly normal (particularly in the three patients with traumatic brain injuries or brain infectious), or the possibility of idiopathic ULD associated with a coincidental lesion, the temporal relationships between the focal lesions herein reported and development of ULD supports a causal link between the two events. The long time interval elapsing in several cases between occurrence of the lesion and emergence of ULD is consistent with the notion that emergence of dystonia may be delayed by months or years following a nervous system insult [43]. Lesion assessment by CT scan could have potentially missed some of the small lesions. However, the results of the study did not change even when considering only patients who performed MRI.

A causal relationships between ULD and the sites of lesion herein reported are biologically plausible. Contralateral ULD in patients with lesions in the basal ganglia, thalamus or frontal cortex may be due to disruption of the basal ganglia motor circuit causing an abnormal input from the thalamus to premotor cortex [44]. In the thalamus, the most commonly affected site was the posterior lateral thalamus, a region that contains relay nuclei receiving afferences from cerebellum/red nucleus and projecting to the cortex [44]. It is worth noting that cerebellar destructive disorders are typically not associated with dystonic phenomena. Consistently, only one patient showed cerebellum damage in our series. Nevertheless, focal dystonia of the hand has been described in cases of degenerative ataxia [45], and a study conducted with positron emission tomography emphasized the participation of the cerebellothalamic pathways in the genesis of dystonic phenomena [46]. The observation that human deep cerebellar stimulation can induce abnormal tonic postures of the neck and limbs would probably imply a pathophysiologic role for increased cerebellar activity rather than loss of activity in some cases [47, 48].

The observation, that brainstem and cervical spine focal lesions were more prominent on the side of the dystonic limb would suggest that the cerebellum and/or its projections could be involved in dystonic disorders associated with damage in these regions. It has been proposed that ULD associated with brainstem or cervical spine lesions might reflect damage of fibers of passage, damage of descending inhibitory supraspinal pathways from reticulospinal tracts, or damage of the pathways linking the basal ganglia, thalamus, and cerebellum to the nuclei where the reticulospinal tracts originate [49, 50]. It has been reported that brainstem lesions involving the medial longitudinal fasciculus, medial reticular formation, red nucleus, and

brachium conjunctivus can produce dystonia in experimental animals [51, 52].

The results of our review on secondary ULD associated with a single focal brain lesion point to several regions, including frontal–parietal cortex, thalamus, basal ganglia, cerebellum, brainstem, and cervical spine. Interestingly, VBM studies in idiopathic ULD have identified GM volume changes in the thalamus, basal ganglia and sensorimotor (frontal/parietal) cortex [6]. Taken together, the studies of idiopathic and secondary ULD support the involvement of multiple brain regions rather than one dominant area in the pathophysiology of ULD. A similar hypothesis has been formulated for other focal dystonias like blepharospasm [53]. In the proposed network model, several brain areas may be involved as "nodes", and ULD may result from dysfunction of one node in the network or also from aberrant communication among the nodes [54].

Task specificity seems to predominate among idiopathic ULD [4], whereas in this sample of secondary ULD task specificity was observed in <8 % of patients. An interesting finding from our analysis was the inverse association between task specificity and thalamic involvement. Owing to the small number of patients with secondary task-specific ULD identified by our review, however, the finding needs to be carefully verified.

In conclusion, our findings support the network model as a likely explanation for the pathogenesis of ULD. Although the presence of dystonic tremor was not associated with any particular site of lesion, the lack of association between task specificity and thalamic involvement would suggest that the various nodes may not be equivalent in terms of the variable clinical expression of ULD.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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